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LITERATURE REVIEW

Whiplash Syndrome

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ABSTRACT

Whiplash syndrome is a quite common pathology and can be defined as a neck injury produced by a sudden acceleration-deceleration, the consequence of which is a sudden forward and backward movement of the head and neck. The main production mechanism is a sudden acceleration-deceleration process that has as a consequence the sudden extension/flexion of the neck. Starting from the many structures involved, the whiplash syndrome is an interdisciplinary challenge (ENT specialist, neurologist, orthopedist, ophthalmologist, psychologist) and can be described by multiple signs and symptoms.

Whiplash syndrome is a complex pathology both through the mechanism of production and symptoms, and through the forensic implications that it has. The interdisciplinary medical collaboration, the implementation of stricter rules on wearing the seat belt and the development by car manufacturers of chairs and head restraints that protect the head and neck of passengers, would be the preventive step in the occurrence and especially the chronicization of these lesions.

KEYWORDS: whiplash, syndrome, acceleration-deceleration, neck pain, dizziness, whiplash-associated lesions.

INTRODUCTION

Described for the first time in 1928, Whiplash syndrome is a rather common pathology and can be defined as the neck injury produced by a sudden acceleration-deceleration whose consequence is a sudden forward and backward movement of the head and neck[1-4]. This type of disorder is associated primarily with road accidents. It is believed that approximately 83% of patients involved in road accidents show the symptoms and signs characteristic of this syndrome.

Besides the term "whiplash syndrome", the term "whiplash-associated lesions" is also described in the literature. The first term refers to lesions produced directly at the level of bone structures and soft tissues of the neck. As for the second term, the derived severe and chronic pathology is included in this name[5].

Whiplash syndrome should be considered a severe condition in terms of the involvement of vital structures at the level of the cervical region, including the bone marrow[6].

PRODUCTION MECHANISM

The main production mechanism is a sudden acceleration-deceleration process that results in sudden neck extension / flexion. The phenomenon occurs especially when the car is hit from behind. At that moment the head and neck are pushed backward into hyperextension, movement immediately followed by forward hyperflexion[6,7]. It is considered that rear impact causes throat hyperextension, while the torso is pushed forward by the car seat and the upper parts of the neck are in a hyperflexion position; this phenomena occurs within fractions of a second, with increased energy, causing the change in the physiological curves of the neck and determining the stretching and injury of both the skeletal system and the soft parts of the neck.

A study with dummies has demonstrated the effect that the impact of two cars has on the passengers in the car hit from behind[8]. While the body remains in place, being held by the seat belt, the neck and head can oscillate in any direction. At the moment of impact, the lower area of the neck – the cervical vertebrae C5, C6 – reaches hyperextension, while the higher cervical vertebrae C2, C3 are in hyperflexion. Thus the cervical spine takes the "S" shape, which is not physiological and seems to cause lesions in the soft tissues of the cervical region, in the peripheral nerves, blood vessels, or even the internal ear[8,9].

The deformation of the cervical region causes

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the elongation of the muscle fiber, the ligaments as well as of nerve structures at the level of the cervical region. Soft tissue lesions, explained by inflammatory mechanisms and translated by the appearance of cervical pain, are associated through cause-effect with the stiffness caused by the contraction of cervical muscles[5,10,11].

At the moment of the forced extension/flexion movement, the peripheral nerves of the cervix can be damaged by the compression and/or elongation phenomenon. Thus, motor injuries, tremor, dystonia can occur[12-15]. An example is the involvement of the brachial plexus; its injury in the whiplash syndrome can be translated as pain and paraesthesia at the level of the upper limb[16].

Approximately 10% of patients with whiplash syndrome experience otological symptoms[17]. The most common are tinnitus, hearing loss and vertigo. These symptoms are thought to be the result of transient ischemia within the labyrinth, as a result of the transient and sudden compression of the vertebral artery, the direct damage to the labyrinth or the acoustic trauma due to the impact noise.

Regarding the vestibular signs and symptoms, there are several theories that underlie their appearance. One thing is certain, the most common type of vestibular deficiency associated with the whiplash syndrome is the benign paroxysmal positional vertigo[18], which is also the most easily treated, but also the one that can be installed immediately after impact or a few days, weeks or months later.

There are authors who support the idea that the instability syndrome occurring after impact is not necessarily due to a vestibular lesion, but due to the antalgic position adopted by the patient in order to avoid neck movement that can cause pain[19,20]. It is known that neck muscles play a particularly important role in maintaining posture[19], aspect that may support the above-mentioned idea.

Another hypothesis is supported by Barre, according to whom cervical lesions would cause the appearance of an irritability syndrome on the sympathetic cervical plexus, a phenomenon that would have the effect of lowering the flow of blood into the labyrinth by vasoconstriction of the internal auditory artery[21]. Hinoki, on the other hand, supports the idea that the overexcitability of cervical proprioceptors and the central nervous system dysfunction (hypothalamus, cerebellum) lead to the imbalance installation[22]. The overexcitability of the sympathetic cervical chain associated with the overexcitability of cervical but also lumbar proprioceptors would be responsible for the appearance of cerebellar symptoms.

Hinoki also shows that hypertonicity of the cervical region muscles involved in maintaining posture is explained by overexcitability both of gamma muscle fibers and of sympathetic nerve fibers at this level[22].

SIGNS AND SYMPTOMS

Starting from the many structures involved, whiplash syndrome is an interdisciplinary challenge (ENT specialist, neurologist, orthopedist, ophthalmologist, psychologist) and can be described by multiple signs and symptoms[2,6,7].

Neck pain and stiffness may occur as a result of muscle and ligament lesions. If pain is described in the anterior cervical region, an injury of the trachea and/or the esophagus should be considered. Normally, these symptoms disappear within 4-7 days after the accident, if the impact was not severe. Otherwise, it may take several weeks.

Dysphagia can also be part of the clinical picture. If the installation is sudden, then we are dealing with a significant sign of pharyngeal edema or retropharyngeal hematoma.

Occipital headache is associated with cervical pain and stiffness.

Another common symptom is pain in the scapular belt, with irradiation on the upper limb, as a sign of a muscle spasm or compression and/or elongation of the nerve trunks (respectively the brachial plexus).

Visual disturbances and nystagmus may occur due to the involvement of the vertebral artery or the sympathetic cervical chain. These symptoms usually occur within a few days or weeks after the accident and improve until extinction in a few months.

From an otological point of view, vertigo, instability and tinnitus are quite common. They usually appear a few days or weeks after the incident and show a tendency to improve in a few months.

In terms of psychic status, the patient may describe anxiety, panic attacks, depression, irritability.

Considering the symptoms and their severity, the lesions associated with the whiplash syndrome can be classified into five degrees according to the Quebec Task Force Classification (Table 1)[5,23].

POSITIVE DIAGNOSIS OF WHIPLASH SYNDROME

The positive diagnosis of the whiplash syndrome and associated lesions raises many difficulties.

In the acute phase, there are a few unspecific signs, which is why the anamnesis is the key element in determining the diagnosis. The discussion

Table 1
Classification of the whiplash syndrome (according to the Quebec Task Force Classification)[5,23]

DEGREE	SYMPTOMS
0	Asymptomatic, without signs of pain or stiffness at the cervical level or other physical signs
I	Cervical pain, stiffness or sensitivity, without physical signs
II	Cervical pain, stiffness or sensitivity associated with musculoskeletal signs (decrease in mobilization capacity and sensitivity threshold)
III	Cervical pain, stiffness or sensitivity and neurological signs (decrease or absence of osteotendinous reflexes, weakness and sensory deficit)
IV	Cervical pain, stiffness or sensitivity and fracture or dislocation

with the patient must establish the pre-existence of a sudden head and neck flexion/extension mechanism. In terms of symptoms, the patient can report immediately after the impact the onset of cervical pain and headache, the rest of signs and symptoms having a later onset.

The clinical examination is also important. Local signs of muscle stiffness, spasm, neurological signs in the upper limb are sought.

The vestibular evaluation is also part of the battery of tests done to the patient. The identification of nystagmus can provide valuable information on the nature of the lesions. It is known that saccades and smooth pursuit are controlled by the central nervous system (CNS), which is why an anomaly in these two parameters may indicate a CNS lesion[24,25].

In assessing the patient with whiplash syndrome, positional tests, caloric tests and computerized dynamic posturography are important from the point of view of the existence of the vestibular lesion and its type[25-28].

Imaging is not indicated in the acute phase, because it can provide many false-positive images by identifying pre-existing degenerative lesions[5,29]. Dynamic radiography of the cervical region can highlight the existence of a kyphotic angle due to the muscle spasm[28]. Both MRI and CT scans can also provide false-positive images. They are recommended when there is a suspicion of cervical spine fracture, ligament lesion and especially if neurological or spinal cord compression signs persist in the long run[5].

TREATMENT OPTIONS

Whiplash syndrome and whiplash-related injuries are difficult to treat in terms of the segments involved, the psychosocial implications, but also the forensic implications.

Prevention is the starting point in the treatment of this pathology, taking into consideration the many forensic cases. Wearing the seat belt, equipping vehicles with efficient head restraints to protect the head and neck, as well as the development and implementation of tests and collaborations between car manufacturers and doctors represent the first step to be taken. Since the whiplash syndrome is most often the consequence of car accidents through rear impact, car manufacturers introduced the head restraint fixed to the car seat, which can be adjusted manually or automatically for each passenger. This limits the movement of the head from hyperextension, limiting the movement differences between the torso and the neck. The correct positioning of the head restraint may reduce the probability of head and neck injuries by 35% during impact.

In the acute phase (less than 4 weeks), when there is subjective cervical pain and paresthesia, but without radiological and clinical signs of cervical spine injury, the neck of the patient can be immobilized with a cervical collar. However, there are studies and guides that do not recommend the use of the collar and show that mobilization and early physiotherapy (up to 96 hours from the incident) can lead to a faster recovery[7,5,30].

The treatment with non-steroidal (NSAIDs) or even steroidal (much less indicated) anti-inflammatory drugs can accelerate the recovery of the cervical region mobilization[5,30].

In the chronic phase (more than 3 months), analgesics may be helpful. Like in the acute phase, multidisciplinary rehabilitation is the key in the treatment of the chronic phase[5,30,31]. The guidelines do not indicate the use of the collar, surgery (especially in asymptomatic cases) or electrotherapy.

CONCLUSIONS

Whiplash syndrome is a complex pathology both through the mechanism of production and symptoms, and by the forensic implications it has.

Early mobilization, physiotherapy from the first days after impact, analgesics (preferably NSAIDs), can contribute to rapid recovery. A symptomatology persisting for more than 3 months shows little chance of recovery.

Although it is not a life-threatening lesion, the "whiplash" disorder involves changes in the quality of life, it may have a chronic evolution and leads to addiction to anti-algic drugs, decreased workplace yield, absence from work, with a strong economic impact.

Interdisciplinary medical collaboration, the implementation of stricter rules on wearing the seat belt and the development by car manufacturers of chairs and head restraints that protect the head and neck of passengers, would be the preventive step in the occurrence and especially the chronicization of these lesions.

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